A STUDY OF UPPER GASTRO INTESTINAL

ENDOSCOPY IN GASTRO INTESTINAL BLEEDING

Dissertation submitted in partial fulfillment of regulation for the award of M.S. Degree in General Surgery

(Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
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Coimbatore - 641 014

CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. R. DEVI SUNDARI** and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The Tamilnadu Dr. M.G.R. Medical University, Chennai

Date: Unit Chief

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DECLARATION

I solemnly declare that the dissertation titled **"A study of upper gastro intestinal endoscopy in gastro intestinal bleeding"** was done by me from 2006 onwards under the guidance and supervision of **Professor Dr. P.M.NANJUNDAPPAN M.S.**

This dissertation is submitted to The Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I).

Place: **Dr. R. DEVI SUNDARI**

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ETHICS COMMITTEE

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Name of the Candidate	:	DR. R. DEVISUNDARI
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Period of Study	:	2006 - 2009
College	:	COIMBATORE MEDICAL COLLEGE
Dissertation Topic	:	A STUDY OF UPPER GI ENDOSCOPY IN GI BLEEDING

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 8.10.2009

N. Neelambika

Secretary
Ethics Committee

ACKNOWLEDGEMENT

Initially I would like to thank the God for guiding me in the right way throughout this work.

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INTRODUCTION

Upper gastrointestinal haemorrhage is one of the important causes of admission in Surgical Intensive care Unit. Despite modern techniques of resuscitation, anesthesia and surgery it has a significant mortality[1]. It was proposed to study the endoscopic evaluation and management of upper Gastrointestinal bleeding in Coimbatore Medical College Hospital , during the period of 2006- 2008, due to the prevalence of UGI (upper gastro intestinal) Bleeding.

Earlier years Barium meal examination[2, 3] had been performed as one of the important diagnostic investigation for acute bleeding. It had two major drawbacks. Erosions and small ulcers cannot be picked up. If a lesion is shown it may not be the actual source of the bleeding.

Gastroscopy had been on use for many years by a few advocates as a visual diagnostic approach .But gastric lesions account only for about a half of all bleeding episodes.

Fibreoptic instruments have recently facilitated and extended the range of examinations. The latest generations are highly flexible and maneuverable ‘panendoscopes’ which allow a complete survey of the esophagus, stomach and duodenum.

Remarkable progress in fibreoptic endoscopy during the last two decades has affected the management of many gastrointestinal disorders. Major technical advances include forward viewing endoscopes with complete tip control and sufficient length to permit direct visualization, of mucosal lesions as far distal as the descending duodenum.

The above facts were the basis for making this study in this institution.

AIM OF THE STUDY

1. To determine the common etiological factors of Upper gastrointestinal bleeding.

2. To establish the site and source of UGI bleeding.

3. To emphasize the value of early endoscopic examination for management in patients with UGI bleeding.

4. To find out the complications and failure rates of endoscopic examinations in patients with UGI bleeding.

REVIEW OF LITERATURE

- Acute GI bleeding is defined as the development of sudden blood loss from the GIT leading to malena, hemetemesis or hemetochezia[4].
- Hemorrhage from the GIT is broadly divided into

A) Bleeding from the upper gastro intestinal tract i.e, proximal to the site of the ligament of Treitz

B) From the lower gastro intestinal tract i.e, distal to the ligament of Treitz

- This generalization has clinical, investigative and therapeutic implications.
- Haemorrhage from the UGI usually manifests as hematemesis or melena and rarely as hemetochezia.

HEMETEMESIS[7, 9]

Defined as vomiting of blood either fresh or digested and altered by gastric secretions. This is a manifestation of a bleeding site located between oropharynx and the ligament of Treitz and may be accompanied by simultaneous malena.The character of the vomitus depends upon the rate of bleeding, the site of bleeding, the rate of gastric emptying. It will be either in the form of coffee ground colour which indicate slower rate

of bleeding with retention in the stomach and formation of acid hematin or fresh blood.

MALENA[6, 9]

It is defined as the passage of black tarry stools. While bleeding sufficient to produce hematemesis usually results in malena, less than half of patients with malena have hemetemesis. Approximately 80 ml of blood is required to produce a single black stool. If there is 1000 ml of bleeding, malena may persist for 3 weeks. The black colour stools secondary to intestinal bleeding results from contact of the blood with hydrochloric acid and also by action of intestinal flora. Characteristically such stools are tarry (sticky).This tarry consistency is in contrast to black or dark stools occurring after the ingestion of iron, bismuth or liquorice.

HEMATOCHEZIA

The passage of bright red blood per rectum generally signifies bleeding from a source distal to the ligament of Treitz. However since blood must remain in the gut for approximately 8 hrs to produce malena, rapid haemorrhage into the esophagus,stomach or duodenum may also result in hematochezia.

CAUSES OF UGI BLEEDING

The most common causes in order of frequency [American society of GI endoscopy]

1.	Duodenal ulcer	24.3%
2.	Gastric erosion	23.4%
3.	Gastric ulcer	21.3%
4.	Varices	10.3%
5.	Mallory weiss syndrome	7.2%
6.	Esophagitis	6.3%
7.	Erosive duodenitis	5.8%
8.	Neoplasm	2.9%
9.	Stomach ulcer	1.8%
10.	Esophageal ulcer	1.7%
11.	Rendu osler weber telengectasia	0.5%
12.	Others	6.3%

Causes enumerated according to the site of origin[7]

It has been tried to enumerate the various causes of upper GI bleeding manifesting either as haemetemesis or malena. For the sake of completion uncommon entities have also been added.

1. Swallowing of blood:
    - Epistaxis
    - Hemoptysis
    - Bleeding from mouth and throat
2. Diseases of the esophagus:
    - Esophageal varices
    - Reflux esophagitis
    - Esophageal ulcer
    - Boerhaave syndrome
    - Corrosive poisoning
    - Epithelioma
    - Aortic aneurysm rupturing into the esophagus
    - Mediastinal growth perforating esophagus
    - Mallory weiss syndrome

3. Diseases of the stomach[5]

- Acute gastritis including drug induced gastritis
- Chronic gastritis
- Corrosive poisons
- Ulcer- Benign and Malignant
- Drugs and gastro intestinal irritants such as Asprin,Arsenic, acids and alkalies, Phosphorous, Phenyl butazone, Corticosteroids, reserpine and indomethacin, Tetracycline, NSAIDS
- Carcinoma
- Leiomyoscarcomas
- Haemorrhagic erosion
- Multiple telangiectasia
- Osler Rendu Weber syndrome
- Hiatus hernia
- Pseudo Xanthoma elasticum(Gron Blad- Strandberg Syndrome)
- Arteriovenous malformations
- Vascular ectasias- water melon stomach(GAVE-Gastric antral vascular Ectasia)
- Dieulafoy's lesion
- Heterotropic pancreas

- Prolapse gastropathy

4. Diseases of the duodenum
    - Ulcer, Duodenitis
    - Diverticula
    - Carcinoma
    - Gall stones
5. Portal obstruction:
    - Cirrohisis of liver
    - Pressure on the portal vein
    - Pylephlebitis
    - Haematobilia
6. Acute febrile diseases
    - HIV
    - Scarlet fever
    - Malaria
    - Yellow fever
    - Dengue fever
    - Cholera
    - Leptospirosis

7. Blood diseases

- Purpura
- Scurvy
- Haemophilia A & B
- Leukemia
- Pernicious anaemia
- Von Willebrand’s disease
- Mastocytosis
- Polycythemia
- Acute erythrocyte sensitivity
- DIC

7. Miscellaneous

- Hemosuccus Uraemia
- Abdominal aneurysm opening into the stomach
- Anticoagulant therapy
- Polyarteritis nodosa
- Malignant hypertension
- Chronic nephritis
- Prolonged diseases
- Syphilis

- Excess strain on the stomach, severe sea sickness
- Abdominal injury
- Vicarious menstruation
- Curling’s ulcer
- Polyps
- Peutz jegher’s syndrome
- pancreatitis

PATHOGENESIS

A) PEPTIC ULCER [10, 25]

Peptic ulcer is the commonest cause of acute upper GI bleeding, accounting to approximately 50% of the cases (Silvastein et al 1981, Gilbert 1990,Rockall et al 1995).

Pathophysiology of peptic ulceration:

Peptic ulceration is due to imbalance between the damaging forces and defence factors.

Damaging Forces

1. Helicobacter pylori
2. Drugs(Corticosteroids, NSAIDS, Aspirin)
3. Acid
4. Pepsinogen
5. Bile

Defence factors;

1. Mucosal blood flow
2. Bicarbanate secretion
3. Prostoglandin

Significant ulcer bleeding is due to erosion of an artery. Bleeding from the ulcer in the posterior wall of the duodenal cap is severe because of erosion of the posterior duodenal artery (Swan et al 1986)

Risk factor for ulceration include

1. Alcohol abuse
2. Drug intake
3. Anticoagulant intake
4. CVS, Pulmonary diseases
5. Liver diseases
6. Smoking
7. Previous renal transplantation
8. States of immune suppression

B. VARICES

A portal venous pressure above 12mmHg and advanced liver diseases are the most important predisposing factors for variceal bleeding.

Causes for PHT(Portal Hypertention) include

1.Cirrhosis

2. Extra hepatic portal vein obstruction

3. Hepatic vein obstruction (Budd Chiari syndrome)

Bleeding from esophageal varices is due to rupture of varices.

1. Erosive theory:

Erosion of the esophageal mucosa can induce variceal bleeding. This can be secondary to NSAIDS, Alcohol, Corticosteroid intake.

2. Transmural tension theory:

Difference in pressure between the esophageal lumen and that of the varix is inversely related to variceal wall thickening.

Laplace's Law $T=TP \times r/w$

TP-Transmural pressure, r-Radius of vessel, w-Wall thickness.

So when the pressure difference increases with the thin variceal wall, bleeding occurs.

3. GASTRIC EROSION:

Frequently associated with NSAID consumption due to inhibition of prostaglandin which is a mucosal protective factor

4. MALLORY WEISS SYNDROME:

Defined as a mucosal laceration of the cardiac or gastroesophageal junction induced by retching or vomiting.

The commonest predisposing factor is alcohol abuse.

Clinical features[13]:

Clinical features of gastro intestinal bleeding depends upon the extent and rate of haemorrhage and the presence of coincidental diseases. Blood loss of less than 500ml rarely produces systemic signs. Exceptions include bleeding in the elderly or in the anaemic patients in whom smaller amounts of blood loss may produce haemodynamic alterations.

The haematocrit and haemoglobin levels are unreliable until equilibration occurs, i.e., six to forty eight hours subsequent to bleeding. Shortly after bleeding has begun an vasovagal reaction occurs which is associated with bradycardia where as with the progression of time the heart rate increases and the cardiac output falls.

Rapid haemorrhage of greater volume results in the fall of venous return to the heart decreasing the cardiac output and increasing the peripheral resistance due to the reflux vasoconstriction.

Orthostatic hypotention, greater than 10mm of Hg usually indicates a 20% greater reduction in blood volume. Concomitant symptoms include syncope, light headedness, nausea, sweating and thirst. Where the blood loss approaches 40% of blood volume, shock frequently ensues with pronounced tachycardia and hypotention.

Pallor is prominent and skin becomes cold and clammy. The clinical picture of shock may reflect a myocardial ischemia or coronary occlusion precipitated by haemorrhage rather than the consequence of massive blood loss per se.

Another consequence of hypotension is decreased renal blood flow resulting in either oliguria or anuria. Azotemia which is characteristically associated with bleeding of esophageal varies also occurs in patients with other types of massive haemorrhage.

Admission risk markers[30, 31] and associated score component values for prediction of need for treatment.

Variable	Admission risk Markers	Score component value
Blood urea(m mol/L)	>6.5<8.0 >8<10.0 >10<25 >25	2 3 4 6
Haemoglobin (g/L) for men	>12<13 >10<12 <10	1 3 6
Haemoglobin (g/L) for women	>10<12 <10	1 6
Systolic Blood pressure(mm Hg)	100-109 90-99 <90	1 2 3
Other markers	Pulse > 100 beats/min Presentation with malena Presentation with syncope Hepatic disease Hepatitis C	1 1 2 2 2

This risk scoring system assess the prediction of need for treatment and assess the outcome and so suitable for use in the clinical setting.

INVESTIGATIONS[16]:

- A)Haemoglobin, blood grouping, Bleeding time, Clotting time, ,Platelets.
- B) Abnormal LFT is an important prognostic indication in bleeding patients.

MODIFIED CHILD PUGH'S CRITERIA[12]:

Nutritional status	Excellent	Good	Poor
Serum bilirubin	<2	2-3	>3
Albumin	3.5	2.8-3.5	<3.5
PT(% of control)	>70	40-70	<70
Ascitis	absent	slight	Moderate
Encephalopathy	none	minimal	Coma

CHILD's grade	A	B	C
Points scored	5-6	7-9	10-15

C) UGI Barium radiography[20, 26]

D) USG & CT

E) UGI endoscopy

F) Angiography

Indication: Endoscopy not revealing the cause

Advantage: Therapeutic embolisation

E) Doppler USG:

1. Demonstrated the anatomy of portal vein, hepatic artery and hepatic vein.
2. Demonstrate porto-systemic shunts and the direction of flow[surgical shunts as well as TIPS].
3. Demonstrate Budd-chiari syndrome.

F) Radionucleotide imaging.

UPPER GASTRO INTESTINAL ENDOSCOPY

UGI ENDOSCOPY [17, 24, 28, 29]

HISTORY OF ENDOSCOPY:

- Early endoscopes were using reflected candle light for illumination.
- 1868, Kussmal performed gastroscopy on a sword swallower, using a rigid endoscope.
- 1886, Mikulicz constructed an open tube rigid esophagoscope which used a miniature light bulb for illumination.
- 1990, Chevalier Jackson modified this into the standard bronchoesophagoscope.
- In the early 1930's, Rudolph Schindler developed a semiflexible gastroscope.
- 1957, Hirschowilz introduced the first flexible fiberoptic gastroscope.
- In1970, the length of the fibroesophagoscope was gradually increased to105 cm so that nearly complete inspection of the oesophagus, stomach and duodenum was possible.
- In 1969 charged couple device (CCD) was introduced in endoscopic instruments.
- In 1980s Welch Allyn introduced electronic endoscope for clinical trial.

UGI VEDIO ENDOSCOPE

PRINCIPLES OF FIBEROPTICS

- The principle of fiber optic light transmission was first patented and developed in Britain, Baird 1928, Hopkins and Kapany 1954. But the development of clinical instrumentation has resulted from technical ingenuits of American and Japanese Engineers.
- Modern endoscopes are flexible because the image is carried by the glass bundles and not by a series of lenses; viewing bundles are 2-4 mm in diameter and consists of many thousands of fine glass fibres.
- Light entering the face of each fibre is transmitted as a series of dots rather like a newspaper photograph.. Its quality depends upon the spatial orientation of the individual fibres, being the same at both ends of the bundle.
- Each fibre is clad with a glass of lower optical density to prevent leakage this clading does not transmit light and is responsible for the fine mesh which is frequently apparent in a fibreoptic image.
- For these reasons the quality of a fiber optic image- although excellent can never equal that obtained with a rigid lens system.

VARICEAL INJECTOR WITH SYRINGE

SCLEROSANT IN USE

SOURCE OF LIGHT:

Bulbs were provided at the tips in either endoscope. Now light is transmitted from an external source through additional fibre bundles. These bundles are randomly arranged.

INSTRUMENTS:

The instrument for upper gastrointestinal endoscopy has a working length of 1 meter and outside diameter of 9-13mm. Distal tip can be deflected up to 200 by a proximal of manual controls. Most of the endoscopes have an image channel, illumination channel and operating channel 2-3mm in diameter for the passage of flexible forceps, cytology brushes and therapeutic devices.

Air insufflation, water cleaning jets and suction are under direct finger tip control. All instruments are designed for photography with a proximal camera. A forward viewing instrument is the best for esophagus and can be passed to the stomach and duodenum. Side viewing instruments are the best for cannulation of Ampulla of Vater and to view certain areas which the forward viewing instrument is blind to. The forward instruments can now be deflected up to 200 degree improving the field.

A compromise is found in certain endoscopes with oblique facing or movable lenses. Trend these days is to have specialized endoscopes for different purposes. Endoscopy can only be performed reliably and safely in a purpose designed or adapted area by properly trained medical and nursing staff.

Methodology and Techniques

1. Endoscopy is performed with the video endoscopic instruments.
2. The patients were kept on empty stomach from previous day 10pm onwards.
3. Ryle's tube aspiration is done in the night and in the morning half an hour before the procedure.
4. 0.012 mg /kg body weight of atropine, 1cc will be given to reduce the secretions. Patient's throat swabbed with 4% xylocaine jelly or 10% xylocaine spray for anesthesia. Injection Diazepam will be given to alley apprehension of the patients.
5. Patient is put on left lateral position and the endoscopy is done under vision past the epiglottis into the oesophagus. The patient was then encouraged to swallow the tube along with the manual guidance and the mucosa was constantly visualized. The instrument should not be passed blindly or undue force should not be used to pass the instrument. Whenever there is "Red out" pull out the instrument and reintroduce the instrument.

A.EXAMINATION OF THE OESOPHAGUS

The mucosa is fully examined and the level of the diaphragm is observed as a slight intentation. The oesophagogastric junction is easy to identify as a change from the slight opaque grey squamous esophageal mucosa to the red glistening gastric folds.

B. EXAMINATION OF THE STOMACH

Air is insufflated into the stomach when the endoscope is at the level of the oesophagogastric junction. The tip is maneuvered slightly downward and to the left initially to obtain a view of the stomach and then upward until the pyloric ring comes into view. The stomach is usually examined completely or withdrawal of the instrument, with particular attention paid to the area just below the angulus on the lesser curve, which is relatively difficult to see but is a common site for ulceration as mentioned previously, complete examination of the fundus of the stomach requires inversion of the endoscope- ‘J ’ maneuver

C.EXAMINATION OF THE DUODENUM

The endoscopist must be convinced that the entire mucosa of the duodenal cap has been visualization. This may be difficult, particularly for the area just distal to the pyloric ring. The mucosa of the cap has a velvety appearance and close examination often reveals small polyps which are usually areas of heterotopic gastric mucosa, Brunner glands or pancreatic rests. This diagnosis can be confirmed by biopsy.

The examination is not complete without attempting to enter the second part of the duodenum which is easily recognized because of the circular muscle folds. Occasionally it may be difficult to exit from the cap. The superior duodenal fold is identified, and the tip is advanced over it, slightly to the right and then strongly downward. At this point withdrawal of the endoscope often results in advancement as the loop in the stomach is straightened.

CLEANING AND DISINFECTION

Instrument is cleaned with soap and water. Biopsy channels are brushed. Endoscope is left in cleaning Solution for 20 min.

2% Gluteraldehyde is the most widely used disinfectant. (Bond et al 1995).

INDICATIONS

DIAGNOSTIC

1. Gastro intestinal bleeding
2. Dyspepsia
3. Heart burns
4. Dysphagia
5. Vomiting

THERAPEUTIC

1. Sclerotherapy in UGI bleeding
2. Banding
3. Dilatation of strictures
4. Stenting in oesophageal malignancy

CONTRAINDICATIONS

1. Anterior osteophytic proliferation of the cervical spine
2. Zenker’s diverticulum
3. Serious systemic disease
4. Recent myocardial infarction
5. Postoperative status

COMPLICATIONS

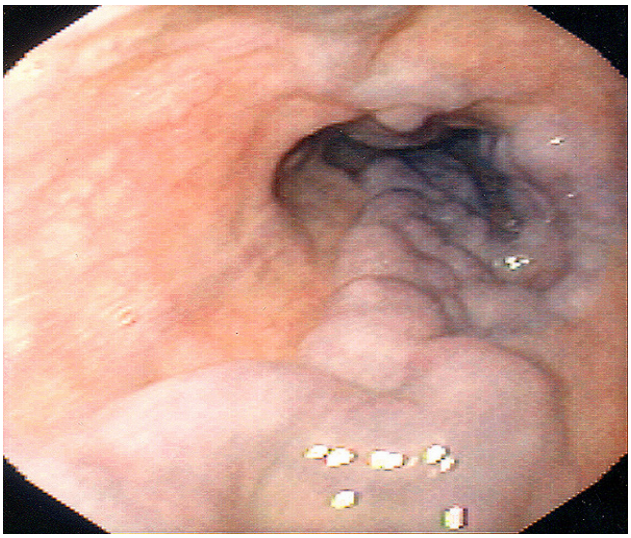
1. Perforation
2. Cardiopulmonary accidents
3. Reactions to medication
4. Infection.

Complications are rare even in the fragile and elderly, perforation in 1 of every 5000 (Schiller and Prout, 1970) seen with inexperienced or therapeutic manipulation like esophageal dilatation even if such occurs can be managed conservatively.

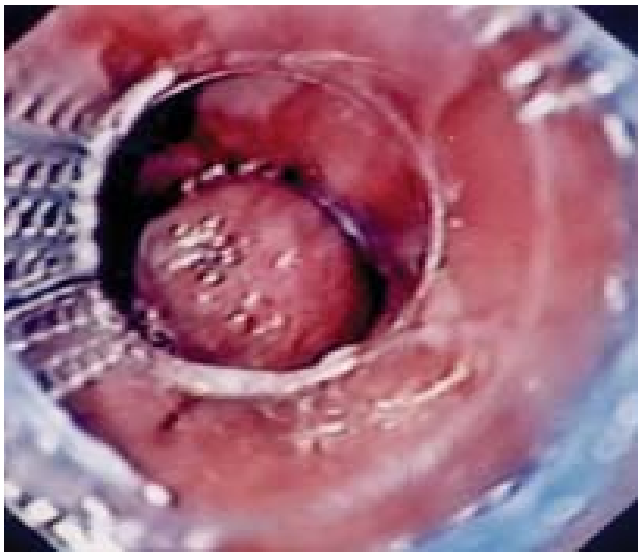
Fatality rate of 1 in 20000 (Schiller and Prout, 1970) has been reported in diagnostic endoscopy. This has resulted from perforation, drug reactions, over sedation and cardiovascular collapse.

At the time where the bleeding spot is sealed with a clot and no further active bleed, one should take precaution to not to dislodge the clot. Patient should be kept under observation apart from general, systemic and supportive measures.

OESOPHAGEAL VARICES



ENDOSCOPIC BANDING



.ENDOSCOPIC FINDINGS[15, 30]

VARICES:

- Endoscopy allows assessment of gastric and esophageal varices. It can also estimate the risk of rebleeding and enable treatment by injection sclerotheraphy or banding.
- Endoscopic grading (By extent of protrusion)

Grade1	<1mm
Grade2	Up to 2mm
Grade3	Up to 3mm
Grade4	>3mm

The largest varices may occupy >1/3 of the lumen.

Predictors of bleeding:

Red color sign:

A portion of the surface of the varix appears red.

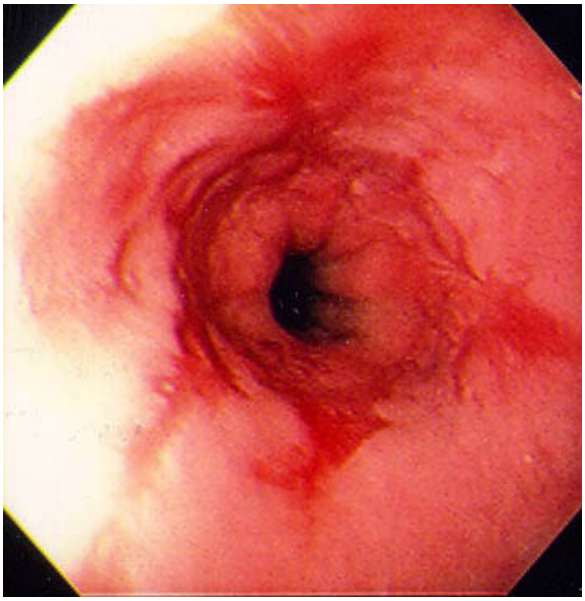
Correlated with risk of rebleeding from 12-52%.

POST BANDING

POST SCLEROTHERAPY

VASCULAR ECTASIA

OESOPHAGITIS



BLEEDING DUODENAL ULCER

HEALED DUODENAL ULCER

BLOCK SPOT(Minor stigma of bleeding ulcer)

ACTIVELY BLEEDING GASTRIC ULCER

Subcategories of Red sign:

1) Red wale sign:

   Dilated venules appearing like red streak on the surface of the varix.

2) Small cherry red spot- <2mm in diameter.

3) A hemotocystic spot- a larger, solitary red spot on the varix

4) A diffuse redness on the area of varix.

B) PEPTIC ULCER DISEASE:

1) Acute PUD:

Single or multiple erosions.

2)Chronic PUD;

Johanson's classification:

Type 1 primary gastric ulcer on the lesser curvature

2 Gastric ulcer with duodenal ulcer

3 prepyloric ulcer

4 High gastric ulcer> 2 cm from oesophageal junction

Matthews and Silen added NSAIDS induced ulcer.

5 They are multiple

C) GASTRIC EROSIONS:

Endoscopy shows acute haemorrhagic gastritis.

MANAGEMENT

The management of haemorrhage consist of 6 phase.

i) Resuscitation –Airway maintenance and O2 administration

-Multiple IV access

-FFP, PL, CP or Packed cell transfusion according to the need

-Balloon tamponade to control bleeding immediately

ii) Diagnosis by endoscopy

iii) Immediate control of bleeding

iv) Prevention of rebleeding

v) Monitoring and discharge

vi) Prevention of recurrent bleeding in the future.

TREATMENT OF BLEEDING PEPTIC ULCER[14, 15]:

Immediate control of bleeding by endoscopy [31]

1) Injection of 0.5 to 1ml of 1 in 1 lakh adrenaline into the ulcer base.
2) Nd YAG Laser
3) Bipolar electro coagulation
4) Injection of sclerosants with or without adrenaline.

Indication for surgery:

1) One severe re bleed
2) Transfusion requirement of 4 U in 24 hrs in a pt over 50 yrs or 6 U in a pt under 50 yrs of age.
3) Failure to localise the bleeding point in someone who is still bleeding severely.
4) Failure to control the sprouting vessel by injection.
5) Aorto gastric fistula.

Surgical procedure:

Bleeding duodenal ulcer- Oversewing the ulcer with a U stitch along with truncal vagotomy and pyloroplasty.

Bleeding Gastric ulcer - Distal gastrectomy with appropriate reconstruction

Medical therapy

1) H2 Antagonist and proton pump inhibitor
2) Helicobacter pylori eradication

Endoscopic therapy for all non variceal UGI bleeding

Thermal method

1) Argon LASER

2) Nd YAG LASER

3) Monopolar electro coagulation

4) Multipolar electro coagulation

5) Heat probe

Injection therapy

1) Adrenaline

2) Sclerosants

a) Polidocanol

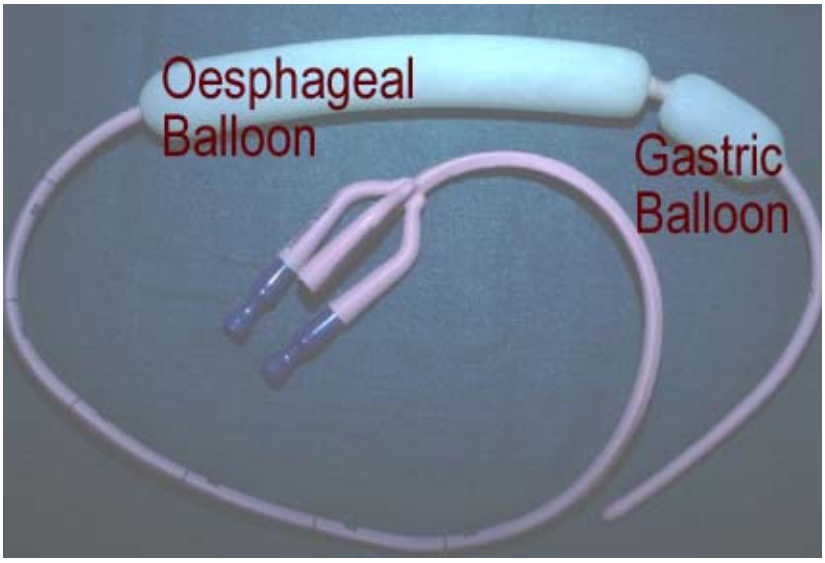
b) Ethanolamine

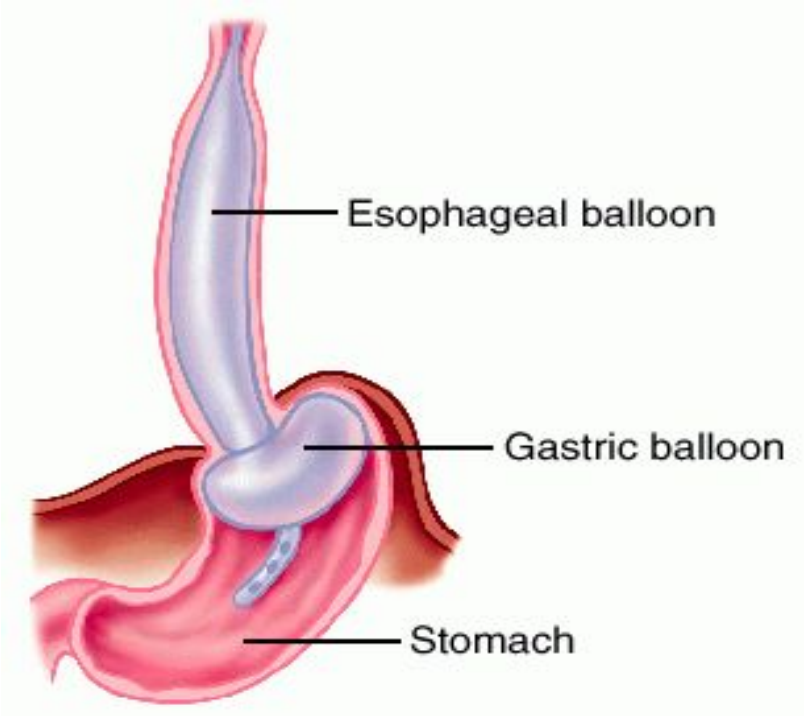
c) Sodium tetradecyl Sulphate

3) Glue-N-butyl cynano acrylate

4) Thrombin

SENGSTAKEN BLAKEMORE TUBE





TREATMENT OF BLEEDING VARICES[30]

1) Resuscitation
2) Pharmacotherapy
    a) Octreotide- 50 microgram iv bolus, followed by 50 –100 microgram per hr infusion
    b) Vasopressin- 20 units in 200 ml of saline over 20 minites followed by 0.1-0.4 unit per min continuous infusion
    c) Propanolol- to prevent recurrent bleeding
    d) Ludothelin- newer epithelial produced factor
3) Endoscopic therapy
    a) Sclerotherapy- intra or parvariceal injection
    b) Endovariceal band ligation
4) Ballon tamponade- using
    a) Sengstaken Blakemore three lumen tube
    b) Minnesota four lumen tube

5) Emergency decompression

TIPS - Transjugular Intrahepatic Porto Systemic Shunt

Before TIPSS

a) Ascitis to be drained

b) Portal/ hepatic vein occlusion to be ruled out

6) Surgical shunt procedures

a) Total shunts- are large diameter shunts which decompress the entire porto mesenteric venous system and divert all portal flow out of or away from the liver

1) End to side porto caval shunts

2) Side to side porto caval shunts

Variants:

1) The portocaval H –graft shunt

2) The mesocaval C-graft shunt-→ very useful procedure in Budd Chiari syndrome

3) Mesorenal shunt

4) Central splenorenal shunt

b) Partial shunts

This shunts decompress the varices while maintaining portal perfusion

c) Selective shunts

Distal splenorenal shunt-Warrant shunt

Coronary caval shunt-Inokuchi shunt

7) Non-shunt procedure

1) Splenectomy
2) Omentopexy, splenopneumopexy
3) Coronary vein ligation
4) Transthorasic ligation of varices
5) Stapled oesophageal transaction
6) Total gastrectomy
7) Hepatic and splenic artery ligation
8) Tanner's procedure-subcardiac portoazygos disconnection
9) Hassab procedure-devascularisation of the upper half of the stomach and oesophagus with splenectomy
10) Splenectomy and coronary vein ligation

CAUSES OF MORTALITY&MORBIDITY[20, 31]

Important cause of death in UGI bleeding is shock and its sequelae.

Comorbid medical illness aggravated due to shock.

Rockall classification to assess risk in acute UGI bleeding:

S.NO	VARIABLE	0	1	2	3
1	Age (yrs)	<60	>60-<79	>80	-
2	Shock (PR in BPM Sys BP in mm Hg)	PR<100 Sys BP >100	PR>100 Sys BP >100	Sys BP<100	-
3	Comorbidity	None	None	Congestive cardiac failure, Carcinoma, Cardiac ischemia	Renal failure,Liver failure, Disseminated carcinoma
4	Diagnosis	Mallory weiss Tear/ no Lesion	All other Diagnosis	Carcinoma of UGI tract Gastro intestinal tract	No lesion idendified
5	Endoscopic Stigmata	Clean base or Dark spot	Blood in GIT	Active bleed/ Adherent clot/ Visible vessel	

MATERIALS AND METHODS

This is a prospective study conducted in the department of General surgery Coimbatore Medical College Hospital , Coimbatore during the period between 2006 to 2008.

The patient admitted in our hospital wards with the history of haematemesis and / or malena have been taken up for the study.

Sample size:

150 cases adult patients with classical UGI bleeding were included in the study.

Inclusion criteria:

1.The patients with history of haematemesis and or malena have been taken up for diagnostic & therapeutic measures.

2.The patients with dyspeptic symptoms and liver diseases have been taken up for diagnostic & preventive measures.

Exclusion criteria:

Children

Method of collection of data:

As soon as the patient is admitted a detailed history regarding the nature of bleeding whether it has ceased at the time of admission and the time since the onset were recorded. The patients were also interrogated regarding symptoms of nausea, vomiting, dysphagia, regurgitation, heart burn, abdominal pain, appetite, wt gain or loss and recent changes in bowel habits prior to the bleed.

A past history of ingestion of drugs over the preceding 48 hrs and frequent ingestion over the preceding months were enquired about and previous history of cardiovascular, respiratory, renal, liver diseases were thoroughly evaluated.

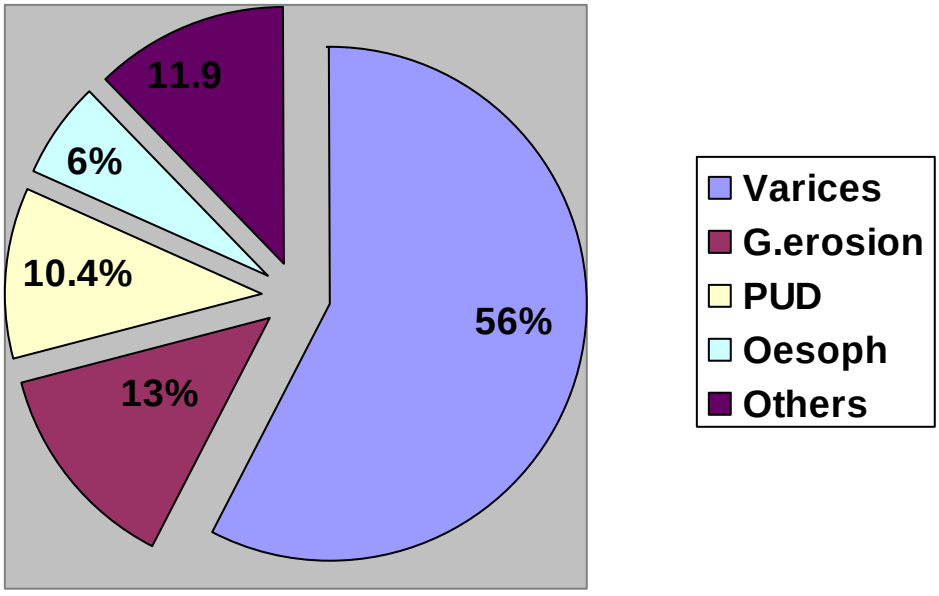
Special emphasis was obviously laid upon the habit of consumption of alcohol by the patient. A detailed examination including patients mental status general appearance and condition of skin were done. Pulse rate, BP, JVP, peripheral edema, signs of cardiac failure were also noted. Examination of the abdomen for any area of tenderness, palpable masses, ascitis and rectal examination were carried out.

Based on clinical data obtained a provisional diagnosis was made. Investigations such as total WBC and differential count, Hb, PCV, BT, CT, urine routine analysis, blood urea, LFT and abdominal scan were done in some cases as and when warranted. These patients were then submitted to oesophagogastro duodenoscopy using a fibreoptic instrument.(PENsTAX-LH-150 P11).The details were entered in a proforma.

ANALYSIS OF CASES

ETIOLOGICAL INCIDENCE (TABLE 1)

S.NO	DISEASE	NO OF CASES	%
1	VARICES	84	56
2	GASTRIC EROSION	20	13
3	DUODENAL ULCER	13	8.6
4	OESOPHAGITIS	9	6
5	GASTRIC ULCER	6	4
6	MALLORY WEISS SYNDROME	3	2
7	MALIGNANCY	2	1.3
8	OTHERS INCLUDING NORMAL STUDY	13	8.6



G. erosion- Gastric erosion **PUD- Peptic ulcer disease**

Oesopho-esophagitis

AGE INCIDENCE (TABLE 2)

S.N	AGE	PHT& VARICES	PUD	EROSIVE GASTRITIS	EOSOPHAGITIS	M.W SYN
1	11-20	9	3	1	-	2
2	21-30	9	4	2	1	-
3	31-40	16	4	3	-	-
4	41-50	22	4	7	3	-
5	51-60	17	1	5	5	-
6	61-70	7	-	2	-	1
7	71-80	4	3	-	-	-

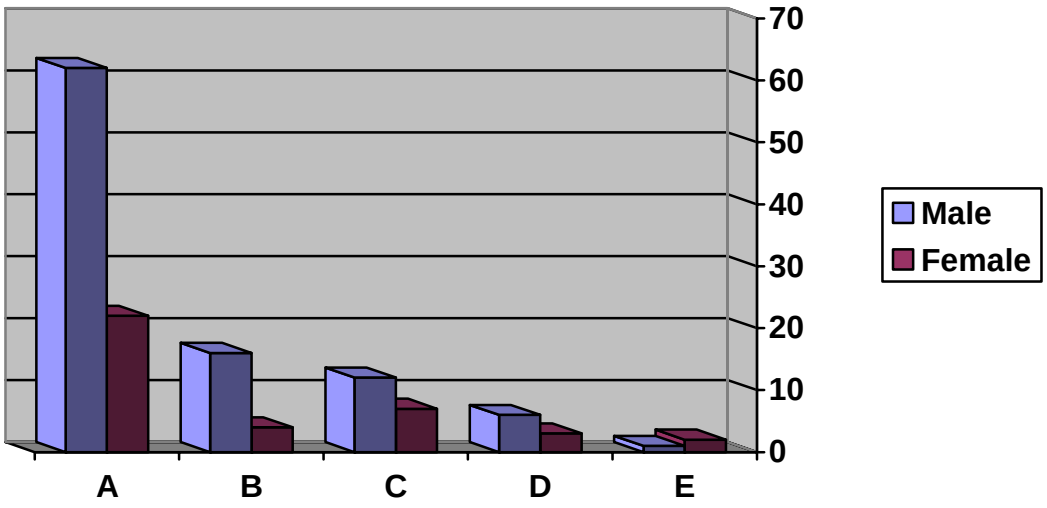
PHT-Portal Hypertention,PUD-Peptic ulcer disease,

M.W.SYN-Mallory weiss syndrome

SEX INCIDENCE (TABLE3)

S.NO	DISEASE	MALE	FEMALE
1	PHT &VARICES	62	22
2	DUODENAL ULCER	9	4
3	EROSIVE GASTRITIS	16	4
4	GASTRIC ULCER	3	3
5	MALLORY WEISS SYNDROME	1	2
6	ESOPHAGITIS	6	3

AGE AND SEX INCIDENCE



A- Varices

B- Erosive gastritis

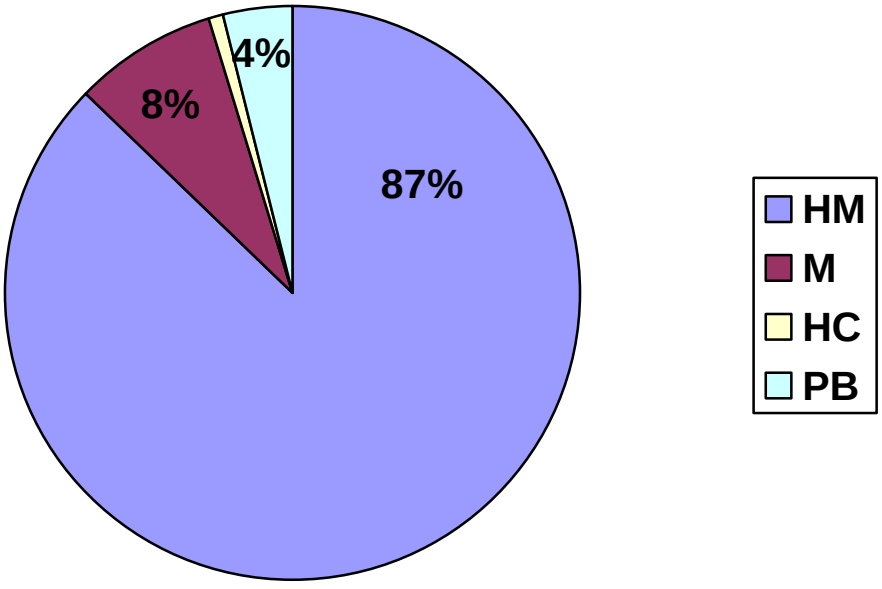
C- Peptic ulcer disease

D- Oesophagitis

E- Mallory weiss syndrome

SYMPTOM ANALYSIS(TABLE 4)

S.NO	SYMPTOM	SYMPTOM ANALYSIS
1	HAEMETEMESIS	131(87.4%)
2	MELENA	12(8%)
3	HAEMATOCHEZIA	1(0.6%)
4	PREVIOUS BLEED	6(4%)



HM- Haemetemesis **HC- Haemetochezia**

PB- Previous bleeding **M- Malena**

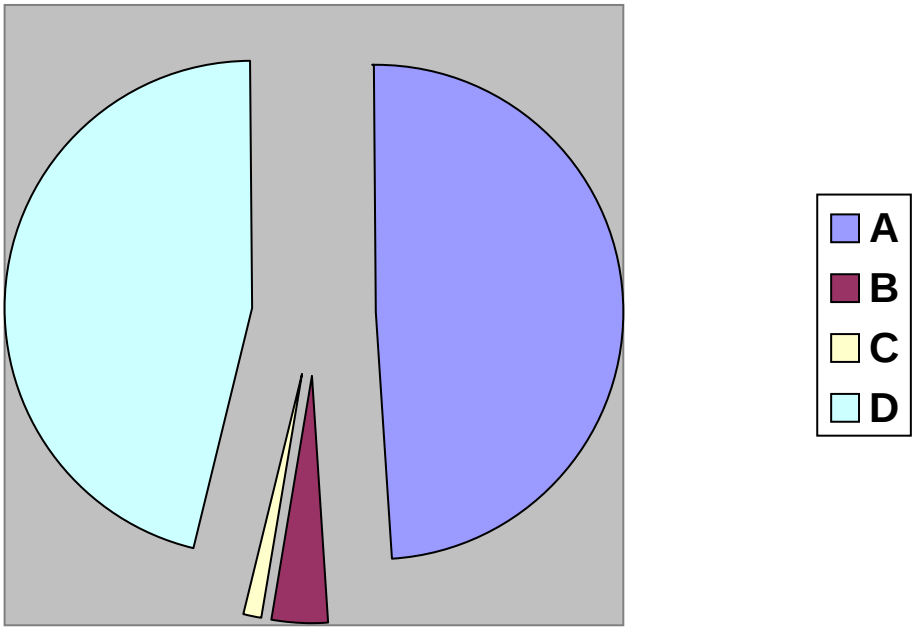
VARICES GRADING(TABLE 5)

S.NO	GRADE	NO OF CASES
1	ISOLATED FUNDAL	2
2	OESOPHAGEAL GRADE—1	6
	GRADE—2	35
	GRADE—3	33
	GRADE-- 4	7
3	COMBINED	5

ETIOLOGY OF PORTAL HYPERTENTION (TABLE 6)

S.NO	ETIOLOGY	%
1.	CIRRHOSIS	41(48.9%)
2.	EXTRA HEPATIC PORTAL HYPERTENTION	3(3.6%)
3.	BUDD CHIARI SYNDROME	1(1.1%)
4.	NOT DETECTED	39(46.4%)

ETIOLOGY OF PORTAL HYPERTENTION



A- Cirrhosis

B- Extra hepatic portal hypertension

C- Budd Chiari syndrome

D- Not detected

TREATMENT OF VARICES(TABLE 7)

S.NO	TREATMENT	NO OF CASES
1	MEDICAL	30(35.7%)
2	ENDOSCOPIC SCLEROTHERAPY	52(61.9%)
3.	BANDING	2(2.4%)
4.	SURGERY(after slerotherapy)	3

One patient was admitted in shock with Sengstaken Blakemore balloon tamponade in situ. After stabilizing the patient endoscopic sclerotherapy was done.

Out of 84 patients with variceal bleeding 61.9% of patients have undergone endoscopic sclerotherapy.

Three patients with recurrent variceal bleeding after sclerotherapy were referred to higher center for surgical management. One of them has undergone splenectomy with lienorenal shunt other two patients have undergone porto systemic shunt with 'H' fistula.

All these three patients are on follow up.

ETIOLOGY&TREATMENT OF EROSIVE GASTRITIS(TABLE 8)

S.NO	FINDING	NO OF CASES
1	ALCOHOL	8
2	DRUG INDUCED	11
3	BOTH	1

All the patients were treated medically and responded well. No surgical treatment was needed.

S.NO	ETIOLOGY	GASTRIC ULCER	DUODENAL ULCER
1	ALCOHOL	4	7
2	DRUG INDUCED	2	6

ETIOLOGY&TREATMENT OF PEPTIC ULCER DISEASE (TABLE 9)

All these patients except one were treated successfully by Antiulcer and Helicobactor pylori eradication therapy.

One male patient with bleeding duodenal ulcer not controlled by endoscopic sclerotherapy was taken up for laparotomy. Oversweing the ulcer was done.

COMPARITIVE STUDIES (TABLE 10)

AUTHORS US SERIES BRITISH SERIES

	Crook et al	Pulmer et al	Katz et al	Schuller et al	Cocks et al	Mailer et al	Avery et al	Cotton et al	This study
Total patients	768	1400	250	2149	2928	87	3936	208	150
Diagnosis (%)									
Varices	11	19	17	2	2	1	-	3	56
Gastric ulcer	18	13	8	15	36	11	17	28	4
Gastric erosion	11	12	20	-	8	8	27	11	13
Duodenal ulcer	42	28	20	29	36	39	35	24	8.6

Esophagitis	-	7	2	-	-	-	-	8	6
No diagnosis	-	7	22	26	4	6	-	15	8.6

DISCUSSION

Upper gastrointestinal bleeding is one of the important cause of admission in surgical Intensive Care Unit requiring critical care and immediate intervention. Upper GI endoscopy is the only useful tool to evaluate all kind of upper GI bleed. Upper GI endoscopy is helpful in identifying the bleeding site and to assess the rate and amount of bleeding. It is handy as a therapeutic interventional device most of the time.

In this study the upper GI endoscopy provided precise and firm diagnosis in 93.3% of patients.

In this study the commonest cause of upper GI bleed was oesophgeal varices of various grade secondary to cirrhosis with portal hypertention.But studies from US , British (table 10) shows that peptic ulcer disease as a commenest cause of UGI bleeding The other causes of portal hypertention include Extra hepatic portal hypertention and Budd- Chiari syndrome.

The common age of affliction of oesophageal varices is between 30-60 yrs. Alcohol is the commonest precipitating factor for cirrhosis of liver and consequently remains the main etiology for majority of upper GI bleed.

Peptic ulcer disease erosive gastritis were also common in male following either consumption of alcohol or ingestion of NSAIDS.

This study shows the value of endoscopy in upper GI bleed as not only a diagnostic tool but also helped in therapeutic intervention especially in patients with variceal bleed. 61.9% of patients with variceal bleeding had successful endoscopic therapeutic sclerotherapy and 2.4% of patients had endoscopic banding. Other patients had been rendered to medical measures.

One patient admitted with shock following variceal bleed had undergone tamponade with Sengstaken Blakemore tube outside in a private hospital was resuscitated with 5 units of blood transfusion and later submitted for upper GI scopy after stabilization. He had grade 3 varices and sclerotherapy was successfully done.

Three patients with recurrent bleed after sclerotherapy were referred for surgical management. One of them has undergone splenectomy

with lienorenal shunt other two patients have undergone porto systemic shunt with ‘H’ fistula.

All these three patients are on follow up.

Most of the non- variceal bleeding in upper GI were amenable to conservative medical management.

One patient who had upper GI bleed was found to have penetrating duodenal ulcer on endoscopy. He was submitted for laparotomy as control of bleeding was not successful. Bleeding was arrested by oversewing the ulcer with a U stitch. Patient recovered after laparotomy.

. Endoscopy was found to be normal in 6.7% of patients inspite upper GI bleed. In all these patients endoscopy was delayed by about 3 days after the last episode of bleeding and probably they had minor gastric erosion,Mallory weiss tear which had healed at the time of endoscopy.

This study highlights the value of endoscopy both as a diagnostic and therapeutic tool in the management of all upper GI bleed.

CONCLUSION

Endoscopy is essential in the initial evaluation and management of upper gastro intestinal bleeding.

In this study UGI endoscopy provided accurate diagnosis in 93.3% of Patients.

The most common cause of UGI bleeding was oesophageal varices.

Therapeutic endoscopy was useful in 36% of patients.

No complications were encountered during / after Endoscopy.

UGI endoscopy serves as the mainstay in the diagnosis of UGI bleeding and its therapeutic apsplications are effective in all cases.

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PROFORMA – I

A Study of the role of upper GI endoscopy in UGI Bleeding

NAME AGE SEX SERIAL NO.

OP/IP NO. ADDRESS OCCUPATION

DOA DOP DOD

1) PRESENTING COMPLAINTS DURATION

a. Haematemesis- quality , quantity
b. Malena-present or absent
c. Jaundice
d. Haematochezia
e. Heart burns
f. Regurgitation- postural, non postural
g. Dyspepsia- recent, chronic

h. Pain abdomen- upper abdomen, anterior chest, back

i. Dysphagia- intermittent, progressive

j. Loss of weight- sudden, slow

k. Loss of appetite

l. Abdominal lump

m. Nausea

n. Vomiting

2) PAST HISTORY

1. DM/HT

2. H/O acid peptic disease

3. H/O intake of NSAIDS,Aspirin

4. Any H/O surgery

5.H/O Sclerotherapy

6. H/O Bleeding diathesis

7.H/O Liver disease

3) PERSONAL HISTORY

Smoking- duration

Consumption of alcohol- duration

Diet- veg/non veg

Betel nut chewing

4) FAMILY HISTORY

Any H/O Liver disease in the family

5) SOCIAL STATUS

6) MARITAL STATUS

GENERAL EXAMINATION

Built/ nourishment

Pallor / icterus/ pedal edema/generalized lymphadenopathy/left supraclavicular node

PULSE RATE BLOOD PRESSURE

ABDOMEN

Tenderness

VGP/ VIP

Dilated veins /scars

Mass/ lump

Hepatomegaly

Ascitis /free fluid

PER RECTAL EXAMINATION

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

CENTERAL NERVOUS SYSTEM

SPINE /CRANIUM

INVESTIGATIONS

1) Blood

Hb, Tc, Dc, LFT, Urea, Sugar and Sr. Creatinine.

2) Stool for occult blood

3) ECG, X-ray chest, X-ray abdomen

4) Barium meal

5) USG Abdomen

6) UGI scopy

DIAGNOSIS

MANAGEMENT AND FOLLOW UP

PROFORMA II ENDOSCOPIC STUDY

NAME AGE SEX GE NO. DATE TIME

SCOPE USED MODEL REFERRED FROM: SURGICAL / MEDICAL

PROVISIONAL DIAGNOSIS PREMEDICATION/ANESTHESIA

ENDOSCOPIC DIAGNOSIS

PROCEDURE ENDOSCOPIST

EMERGENCY ASSISTANT

ROUTINE STAFF

FOLLOW UP

THERAPUETIC POST OP

FINDINGS

OESOPHAGUS

OESOPHAGO GASTRIC JN

STOMACH

DUODENUM

ENDOSCOPIC PICTURES TAKEN: YES/ NO

SPECIMEN TAKEN: YES/ NO

BRUSH CYTOLOGY

FORCEPS BIOPSY

SITE OF BIOPSY

PATHOLOGY

STUDY

i. COMPLETE / INCOMPLETE
ii. NORMAL/ ABNORMAL
iii. NEXT FOLLOW UP NEEDED / NOT NEEDED
iv. FINAL DIAGNOSIS

SITE OF LESION

ADVICE - MEDICAL

SURGICAL

COMBINATION

MASTER CHART

S.No	NAME	AGE/ SEX	GE. No	S/A	DRUG	CLINICAL FEATURES	ENDOSCOPIC, FINDINGS & MANAGEMENT
1	Ravi	45/M	1697/07	A	-	Pallor,H	G 3 oesophageal Varices, 3 coloums, Distal 5 cm, EST done
2	Kannappan	50/M	1670/07	S	-	H	Oesophagitis
3	Kanniammal	36/F	1816/07	S	-	H	Normal
4	Jothimani	45/F	1813/07	A	-	H,Sp,As	G 2 varices, 3 coloums, distal 7cms. Oesophagitis
5	Marimuth	68/F	1850/07	A,S	-	H	Ulceroproliferative growth at 20cms of oesophagus
6	Selvi	34/F	1848/07	A	-	H,Sp,As	G 3 varices, 4 coloumns, distal 9 cm, EST done
7	Subbaiyan	65/M	1909/07	A,S	+	H	Gastric erosion
8	Jayamal	49/F	2019/07	-	-	H	Normal
9	Ravikumar	40/M	1992/07	A	-	H	G 2 oesophageal varices,3 col, distal 6 cms
10	Kanimozhi	30/F	1985/07	A	-	H	Normal
11	Arul das	57/M	2037/07	A,S	+	H	Diffuse gastric erosion
12	Mutulaxmi	44/F	2073/07	A	+	H	Gastric ulcer
13	Vijaya	48/F	2149/07	-	-	H	Oesophagitis
14	Chinnasamy	64/M	1380/07	A,S	-	H,Sp,As	G 2 varices, 3 coloums, Distal 6cm, EST done.
15	Veeramal	45/F	2220/07	A,S	-	H	Large prepyloric ulcer
16	Vijaya	48/F	2149/07	A	+	H	Gastric erosion
17	Karupusamy	56/M	2223/07	A,S	+	H	Gastric erosion
18	Kamalam	56/F	2242/07	A	-	H,Sp,As	G 3 oesophageal, Varices
19	Kalimuthu	55/M	2245/07	-	+	H	Normal
20	Rajan	57/M	2253/07	A	-	H,Sp	G 2 oesophageal, Varices
21	Thangapan	72/M	2284/07	A	-	H	G 2 oesophageal, Varices
22	Marappan	73/M	2284/07	A	-	H	G 2 oesophageal varices
23	Muthammal	48/F	2332/07	A,S	+	Hematochezia	Ulcer with active ooze from duodenal bulb,Laporotomy done
24	Chinathambi	17/M	2115/07	A,S	-	H,Sp,As	G3 oesophageal varices, 4 col, distal 9cms, EST done
25	Dhanalxmi	57/F	2448/07	A	-	H	G1 oesophageal varices, 2 col, EST done.

26	Krishnan	60/M	2563/07	A	-	H	Normal
27	Sengodan	65/M	2566/07	A,S	-	M	Gastric erosion
28	Malar	18/F	2582/07	A,S	+	H	Active bleed from ulcer in greater curvature
29	Vairamani	15/M	863/07	A	-	H,EHPVO	G3 oesophageal varices
30	Velusamy	60/M	291/08	-	-	H	Normal
31	Ramasamy	39/M	22/08	A	-	H,Sp	G2 oesophageal varices
32	Balakumar	15/M	1904/02	A,S	-	H	G2 oesophageal varices, EST done
33	Kuppan	73/M	83/08	A,S	-	H,Budd chiari Syndrome	G3,4 varices, 3 col,Distal 10 cms, EST done
34	Nabees	15/M	147/08	A,S	-	H	G2 varices, 4 col, distal 6 cms, EST done
35	Nataraj	52/M	167/08	A,S	-	H	G2 oesophageal varices, 3 col, EST done
36	Jeganathan	71/M	175/08	A	-	H,Sp	G2 oesophageal varices
38	Sanmugaraj	21/M	192/08	A	-	H,EHPVO,As	G3 oesophageal varices,EST done,Splenorenal shunt done.
39	Ponnusamy	57/M	200/08	A,S	-	H	G2 oesophageal varices, EST done
40	Sukuraj	40/M	247/08	-	+	H	Normal
41	Palaniamal	75/F	237/08	A,S	+	H	Gastric erosion
42	Rajan basu	60/M	235/08	A,S	-	H,As,Sp	G2 varices, 3 col, distal 7 cms, EST done. congestive gastropathy
43	Sekar	31/M	256/08	A	-	H,M	G3 oesophageal varices , EST done
44	Ravi kumar	38/M	1992/07	A	-	H	G3 varices,3 col
45	Kandasamy	47/M	92/08	A	-	H,Sp	G3 varices, 3 col distal 8 cms
46	Arulraj	58/M	292/08	A	-	H	G1 varices, EST done
47	Balesh	34/M	2319/06	A	-	H	G3 varices, 3 col, EST done
48	Ravikumar	32/M	1992/07	A	-	H	G3 varices,3 col, EST done
49	Selvaraj	54/M	333/08	A,S	-	H	Active DU
50	Rajendran	47/M	346/08	A	-	H	G1 oeso varices EST done
51	Chidambram	62/M	340/08	A	-	H,Sp	G3 varices,3col, EST done

52	Srirangam	47/M	405/08	A	-	H	G2,oesophageal varices
53	Muniappan	46/M	203/08	A	-	H	G2, oesophageal varices
54	Sigamani	42/M	426/08	A	-	H	G2, oesophageal varices, 3 col
55	Rajamani	70/F	444/08	A	-	H,Sp	G2 oesophageal varices, 3 col
56	Prabhu	24/M	317/08	A	+	H	Gastritic erosion
57	Vijayalaksmi	55/F	952/07	A	-	H	Fundal varices
58	Chandran	52/M	460/08	S	-	H	DU,2nd part
59	Selvam	30/M	517/08	A,S		H,M	DU,Bulb.
60	Velusamy	70/M	555/08	-	+	H	Normal
61	Nazeer	45/M	597/08	A	-	H	Normal
62	Parvathy	60/F	2051/06	A	-	H,As	G3, oesophageal varices
63	Raveendren	68/M	2015/06	A,S	-	H	G3, oesophageal varices
64	Indirani	50/F	1983/06	A	-	M,As	G3, oesophageal varices, EST done
65	Ayyavu	58/M	2101/06	-	-	H	Oesophagitis
66	Dhanam	42/F	2135/06	A,S	-	H	Gastric erosion
67	Rajini	53/F	163/06	-	-	H	Oesophagitis
68	Karupusamy	60/M	2140/06	-	+	H	Gastric erosion
69	Palanisamy	71/M	457/07	A	-	H	DU
70	Muralidaran	35/M	2167/06	S	+	H	Gastric ulcer
71	Arumugam	30/M	2223/06	-	-	H	0sophagitis
72	Subashini	14/F	2224/06	A,S	-	H	Gastric erosion
73	Krishnaveni	54/F	2189/06	A,S	-	H	G2, oesophageal varices
74	Sentram	63/F	2188/06	A,S	-	H	External compression 0f stomach
75	Joice	13/F	2227/06	A	-	H	Mallory Weiss tear
76	Akbar	53/M	2217/06	S	-	H	Oesophagitis
77	Karupusamy	24/M	2226/06	A	-	H,Sp	G3, oesophageal varices,Surgery(H-shunt)done
78	Chandramohan	42/M	1122/06	A	-	H	G2, oesophageal varices
79	Murugesh	49/M	3168/06	A,S	+	H	Gastric erosion
80	Ranganathan	47/M	2220/06	A	-	H	Gastric erosion
81	Sivadoss	42/M	2248/06	A	-	H	G1, oesophageal varices,Banding done
82	Nithya	15/F	2256/06	A	-	M	G2, oesophageal varices

83	Mohan	52/M	1122/06	A,S	-	H,As,Sp	G3, oesophageal varices, EST done
84	Sekar	42/M	2207/06	A	-	H	G3, oesophageal varices
85	Rajan	44/M	2266/06	A,S	-	H	G3, oesophageal varices, 2 col, Banding done
86	Kaliammal	74/F	2314/06	A	-	H	G2, oesophageal varices, 2 col, EST done
87	Paleesh	30/M	2319/06	A	-	H,As	G2, oesophageal varices 3 col, EST done
88	Bagya raj	37/M	2569/06	A,S	-	H,Sp,As	G4, oesophageal varices, 4 col, EST done
89	Pattusamy	57/M	2089/06	A	+	H	Erosion at the site cystogastrotomy
90	Perumal	47/M	2518/06	A	-	H	G1, oesophageal varices
91	Ravi	42/M	2532/06	A	-	H	Gastric erosion
92	Charulatha	19/F	2479/06	A	-	H	Mallory Weiss tear
93	Koteeswaran	22/M	2696/06	S	-	H	DU
94	Ravi	49/M	2718/06	A	-	H,As	G3, oesophageal varices, 4 col EST done
95	Marimuthu	18/M	2735/06	A	-	H,M	G2,3, oesophageal varices, 3 col EST done
96	Selva raj	32/M	2759/06	A	-	H,Sp	G4, oesophageal varices, 4 col EST done
97	Mangammal	72/F	2782/06	-	-	H	Osophagitis
98	Srirangam	55/M	2815/06	A	-	H	DU
99	Dhanapal	35/F	41/07	-	-	H,As,Sp, EHPVO	G4, oesophageal varices,1 col
100	Lakshmi	50/F	23/07	A,S	-	H,M	G2, oesophageal varices ,3 col, EST done
101	Mani	40/M	526/06	A	-	H	G1 oesophageal varices
102	Nanjammal	41/F	274/06	A	-	H,Sp,As	G3, oesophageal varices, 3 col, EST done
103	Anwar batsa	87/M	87/07	-	+	H	Gastric erosion
104	Jothi	30/F	233/07	A,S	-	H,A	G3, oesophageal varices, 4 col, EST done
105	Pachiammal	67/F	228/07	A	-	H	G3,oesophageal varices, 3 col, EST done
106	Kajamoideen	34/M	165/07	S	-	H	DU
107	Kumar	18/M	249/07	A	-	H,Sp	G3, oesophageal varices, 3 col, EST done
108	Krishnamal	45/F	245/07	A	-	H,As	G3, oesophageal varices, 3 col ,EST done
109	Eswari	67/F	303/07	A	-	H	G3, oesophageal varices, EST done
110	Thangavelu	47/M	372/07	A	-	H	Oesophagitis
111	Arpudasamy	57/M	390/07	A,S	+	H	Gastric ulcer
112	Parameswari	52/F	393/07	A	-	M,Sp	G3, oesophageal varices

113	Raja	29/M	474/07	A	-	H,Sp	G3,oesohageal varices
114	Selvaraj	32/M	275/07	A	-	H	G4, oesophageal varices, EST done,Surgery done(H-shunt)
115	Palanisamy	40/M	542/07	-	+	H	Gastric erosion
116	Rathnasamy	56/M	578/07	A	-	H	DU
117	Maheswari	23/F	566/07	A	-	H	G2 oesophageal Varices
118	Siva	42/M	224/07	A,S	-	H,M	Rebleeding oesphagealvarices-EST done
119	Pichiammal	31/F	567/07	A	-	H,HBSAg+, As,Sp	G3 oesophgeal varices
120	Ponnusamy	52/M	591/07	A	-	H	G2 Oesophageal varices
121	Vijayakumar	34/M	590/07	A	+	H	Gastric ulcer
122	Boopathy	31/M	654/07	A	-	H	DU
123	Bazeer	36/M	657/07	A,S	-	H	G2 oesophageal varices, 2 col
124	Masillamani	42/M	578/07	A,S	-	H,Sp,As	G3 oesophageal varices, 4 col, EST done
125	Palanisamy	64/M	690/07	A,S	-	H	G3, G4 oesophageal varices, 4 col, EST done
126	Abdulsamed	52/M	647/07	A	-	H	Fundal varices
127	Subbathal	60/F	693/07	A,S	-	H	G2 oesophageal varices, 3 col, EST done
128	Selvam	27/M	726/07	A	-	H,Sp	G2 3 col ,EST done
129	Krishna	64/M	755/07	A	-	H	Mallory Weiss tear
130	Balan	64/M	781/07	A,S	+	H,M	Growth in the body of stomach
131	Kanagaraj	55/M	812/07	A	-	H	G2 oesophageal varices, 2 col, EST done
132	Sarojini	54/F	883/707	A	-	H,Sp	G3 oesophageal varices, 3 col ,EST done
133	Indirani	58/F	643/07	-	+	H	Gastric erosion
134	Karpagaveni	18/F	1045/07	A	-	H	DU
135	Seetha	45/F	1181/07	A,S	-	H,As,Sp	G2, G3 oesophageal varices, 3 col, EST done
136	Lakshmi	67/F	1228/07	A	-	H,Sp	G3 oesophageal varices,4col,EST done
137	Karupusamy	50/M	1224/07	A	+	H	Active DU
138	Kalyani	23/F	1285/07	A,S	-	H	G3 oesophageal varices,EST done
139	Ammasai	42/M	1425/07	A	-	H,Sp	G2,0esophageal varices
140	Selvam	27/M	726/08	A	-	H	G2 0esophageal varices
141	Chinnaiyan	53/M	1476/08	A	-	H,Sp	G2 Oesophageal varices,3col,EST done
142	Chinathambi	17/M	56/08	A	-	M,As,Sp	G3 oesophageal varices, 4 col, EST done

143	Vairamani	25/M	68/08	-	-	H	Oesophagitis
144	Yoganya	15/F	89/08	S	+	H	Gastric erosion
145	Venkat	52/M	134/08	A	-	H	DU
146	Kitturaj	33/M	165/08	A,S	-	H	Gastric erosion
147	Ravikumar	28/M	256/08	A,S	-	H	DU
148	Mylathal	56/F	289/08	A	-	H	Normal
149	Santhosh	37/M	368/08	A,S	+	H	Gastric erosion
150	Sakthi	28/M	430/08	-	-	H	DU

A-Alcoholism, S-Smoking, H-Hemetemesis, M-Malena, As-Ascitis, Sp-Splenomegaly,

DU-Duodenal ulcer, EST- Endoscopic Sclerotherapy, G-Grade.